The Marr and Albus theories of the cerebellum
Two early models of associative memory

by

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Abstract

The Marr and Albus theories of the cerebellum are compared and contrasted. They are shown to be similar in their analysis of the function of the mossy fibers, granule cells, Golgi cells, and Purkinje cells. They both predict motor learning in the parallel fiber synapses on the Purkinje dendrites mediated by concurrent climbing fiber input. This prediction has been confirmed by experimental evidence. In contrast, Marr predicts these synapses would be facilitated by learning, while Albus predicts they would be weakened. Experimental evidence confirms synaptic weakening.

Introduction

Two papers published in 1969 and 1971 by David Marr and James Albus form the basis for what has become known as the Marr-Albus theory of the cerebellum.
Both of these papers were inspired by, and draw most of their data from, a book by Eccles, Ito, and Szentagothai entitled The Cerebellum as a Neuronal Machine.[Eccles67]

"A diagram of the general cerebellar cortical structure appears in Fig. 1. The cortex has two types of afferent fiber, the climbing fibers (Cl) and the mossy fibers (Mo). Each climbing fiber makes extensive synaptic contact with the dendritic tree of a single Purkinje cell (p), and its effect there is powerfully excitatory. The axons of the Purkinje cells leave the cortex (they form the only cortical output) and synapse with cells of the cerebellar nuclei.

"The second input, the mossy fibers, synapse in the cerebellar glomeruli (gl) with the granule cells. Each glomerulus contains one mossy fiber terminal (called a rosette), and dendrites (called claws) from many granule cells. The glomerulus thus achieves a considerable divergence, and each mossy fiber has many rosettes."

"The axons of the granule cells rise (g) and become the parallel fibers, which synapse in particular with the Purkinje cells whose dendritic trees they cross. Where the granule cell axons (i.e. the parallel fibers) make synapses, they are excitatory.
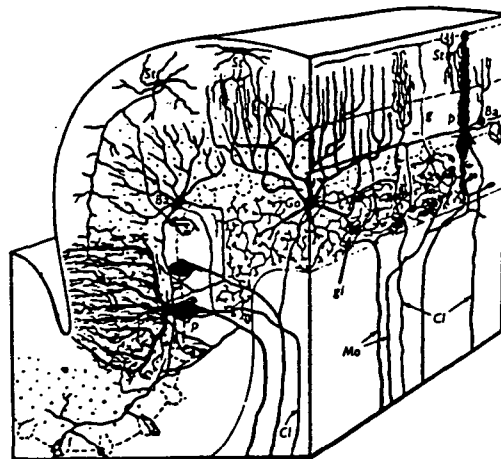
"Fig. 1. Diagram of cerebellar cortex (from Eccles et al. 1967, Fig. 1). The afferents are the climbing fibers (Cl) and the mossy fibers (Mo). Each climbing fiber synapses with one Purkinje cell (p), and sends weak collaterals to other cells of the cortex. The mossy fibers synapse in the cerebellar glomeruli (gl) with the granule cells whose axons (g) form the parallel fibers. The parallel fibers are excitatory and run longitudinally down the folium: they synapse with the Purkinje cells and with the various inhibitory interneurones, stellate (St), basket (Ba) and Golgi cells (Go). The stellate and basket cell axons synapse with the Purkinje cells, and the Golgi cell axons synapse in the glomeruli with the granule cells. As well as their ascending dendrites, the Golgi cells possess a system of descending dendrites, with which the mossy fibers synapse in the glomeruli. The Purkinje cell axons form the only output from the cortex, and give off many fine collaterals to the various inhibitory interneurones."

"The remaining cells of the cortex are inhibitory interneurones. The Golgi cells (Go) are large, and have two dendritic trees. The upper tree extends through the molecular layer, and is driven by the parallel fibers. The lower dendrites terminate in the glomeruli, and so are driven by the mossy fibers. The Golgi axon descends and ramifies profusely: it terminates in the glomeruli, thereby inhibiting the granule cells. Every glomerulus receives a Golgi axon, almost always from just one Golgi cell: and each Golgi cell sends an axon to all the glomeruli in its region of the cortex.

"The other inhibitory neurones are stellate cells, the basket (Ba) and outer stellate (St) cells. These have dendrites in the molecular layer, and are driven by the parallel fibers. Both types of cell synapse exclusively with Purkinje cells, and are powerfully inhibitory.

"Finally, the cortex contains various axon collaterals. The climbing fibers give off weak excitatory collaterals which make synapses with the inhibitory interneurones situated near the parent climbing fiber. The Purkinje cell axons give off collaterals which make weak inhibitory synapses with the cortical inhibitory interneurones, and perhaps also very weak inhibitory synapses with other Purkinje cells. These collaterals have a rather widespread ramification.

"Behind this general structure lie some relatively fixed numerical relations. These all appear in Eccles et al. (1967), but are dispersed therein. It is therefore convenient to set them down here.

"Each Purkinje cell has about 200,000 (spine) synapses with the parallel fibers crossing its dendritic tree, and almost every such parallel fiber makes a synaptic contact. The length of each parallel fiber is 2-3 mm (1 1/2 mm each way), and in 1 mm down a folium, a parallel fiber passes about 150 Purkinje cells. Eccles et al. (1967) are certain each fiber makes at least 300 (of the possible 450) synaptic contacts with Purkinje cells, and think the true number is nearer 450. There is one Golgi cell per 9 or 10 Purkinje cells, and its axon synapses (in glomeruli) with all the granule cells in that region, i.e. around 4500. There are many granule cells (2.4×10^6 per mm of granule cell layer), each with (usually) 3-5 dendrites (called claws): the average is 4.5 and the range 1-7. Each dendrite goes to one and only one glomerulus, where it meets one mossy fiber rosette. It is,

however, not alone: each glomerulus sees the termination of about 20 granule cell dendrites, possibly a Golgi cell descending dendrite, and certainly some Golgi axon terminals, all from the same Golgi cell. Within each folium, each mossy fiber forms 20-30 rosettes, giving a divergence of 1 mossy fiber to 400-600 granule cells within a folium. The mossy fiber often has branches running to other folia.

"Just below the Purkinje cells are the Golgi cell bodies, and just above them are the basket cell bodies. There are 10-12% more basket cells than Purkinje cells, and about the same number of outer stellate cells. Each basket cell axon runs for about 1 mm transversely, which is about the distance of 10 Purkinje cells. The basket axon is liable to form baskets round cells up to three away from its principal axis, so its influence is confined to a sort of box of Purkinje cells about 10 long and 7 across. The distribution of the outer stellate axons is similar except that it has a box about 9 x 7, since its axon only travels about 0.9 mm transfolially. The outer stellates inhabit the outer half of the molecular layer, and the basket cells the inner third. There are intermediate forms in the missing sixth. None of these cells has a dendritic tree as magnificent as that of the Purkinje cell, and Eccles et al. (1967) do not venture any comparative figures. Some outer stellates are small, with a local axonal distribution. A lot of the synapses of parallel fibers with this last group of cells are directly axo-dendrite, but all other parallel fiber synapses are via spines, though these are of different shapes on the different sorts of cell. Calculations based on slightly tenuous assumptions suggest that each Purkinje cell receives connections from about 7000 mossy fibres." [From Marr 1969]

Both Marr and Albus agree on the nature and function of the mossy fibers, granule cells, and Golgi cells, i.e. that they recode input patterns of mossy fiber firing rates into patterns of parallel fiber activity.

Marr expresses the recoding in terms of codons.

"The synaptic arrangement of the mossy fibers and the granule cells may be regarded as a device to represent activity in a collection of mossy fibers by elements each of which corresponds to a small subset of active mossy fibers. It is convenient to introduce the following terms: a codon is a subset of a collection of active mossy fibers. The

representation of a mossy fiber input by a sample of such subsets is called the codon representation n of that input: and a codon cell is a cell which is fired by a codon. The granule cells will be identified as codon cells, so these two terms will to some extent be interchangeable. The size of codon that can fire a given granule cell depends upon the threshold of that cell, and may vary: and the mossy fibers which synapse with the granule cell determine the codons which may fire that cell.

"There are exactly $\binom{L}{R} = \frac{L!}{R!(L-R)!}$ codons of size R associated with a collection of L active mossy fibers. If two mossy fiber inputs each involve activity in L fibers of which W were common to the two, the two inputs are said to overlap by W elements; and they may be expected to have some codons in common. In fact the number they share is precisely $\binom{W}{R}$. The ratio X of the number of shared codons to the number of codons each possesses is given by $X = \binom{W}{R}/\binom{L}{R} = \frac{W(W-1)...(W-R+1)}{L(L-1)...(L-R+1)}$

which tends to (W/L)^R as W increases. The limiting values of X for relevant values of R appear in Table 1. It will be observed that the effect of the subset coding is to separate patterns, because similar inputs have markedly less similar codons.

TABLE 1. Overlap Table, i.e. values of $(W/L)^R$

(W/L)	R = 2	3	4	5
0·5	0·25	0·12	0·06	0·03
0·6	0·36	0·22	0·13	0·08
0·7	0·49	0·34	0·24	0·17
0·8	0·64	0·51	0·41	0·33
0·9	0·81	0·73	0·66	0·59

"The mossy fiber granule cell relay effectively takes a sample of the codon distribution of an input: the sample is small enough to be manageable, but large enough for the input event to be recoverable from it with high probability." [From Marr 1969]

Marr's concept of codons derives from Brindley [Bri69], and is elaborated in later papers by Marr. From analysis of codon theory, Marr predicts that the number of responses that can be stored by each Purkinje cell is less than 500, and probably around 200.

Albus expresses the recoding in terms of Perceptron theory [Ros61].

"Assume a decoder, or rather a recoder, that codes N input fibers (mossy fibers) onto 100N association cells (granule cells). Such a recoding scheme provides such redundancy that severe restrictions can be applied to the 100N association cells without loss of information capacity. For example, it is possible to require that of the 100N association cells, only 1% (or less) of them are allowed to be active for any input pattern. That such a recoding is possible without loss of information capacity is easily proven, for 2^N is much smaller than 100^N things taken N at a time.

"That such a recoding increases the pattern-recognition capabilities of a Perceptron is certain, since the dimensions of the decision hyperspace have been expanded 100 times. The amount of this increase under conditions likely to exist in the nervous system is not easy to determine, but it may be enormous. It can be shown that 100N things taken N at a time is greater than 100^N. Thus 2^N possible input patterns can be mapped very sparsely onto 100^N possible association cell patterns. If this is done randomly, the association cell patterns are likely to be highly dissimilar and thus easily recognizable. The ratio 100^N/2^N = 50^N rapidly increases as N becomes large.

"The restriction that only 1% of the association cells are allowed to be active for any input pattern means that any association cell participates in only 1% of all classifications. Thus its weight needs adjusting very seldom and there is a fairly good probability that its first adjustment is at least in the proper direction. This leads to rapid learning." [From Albus 1971]

From analysis of Perceptron theory, Albus predicts that the number of patterns that can be recognized by each Purkinje cell is on the order of 200,000.

The large difference between Marr and Albus in predicting Purkinje discrimination capacity is due to differences in the hypothesized mechanism of learning. Marr suggests that learning takes place only by facilitation of positive synaptic weights between parallel fibers and Purkinje dendrites. Albus suggests a mechanism by which synaptic influence can effectively be adjusted in both positive and negative directions. This is accomplished through modification of parallel fiber synapses not only on Purkinje dendrites, but on Basket and Stellate b cells as well.

Marr and Albus agree in suggesting that climbing fibers control cerebellar learning by modification of synaptic weights between parallel fibers and Purkinje dendrites. There is, however, a

significant difference between Albus and Marr regarding the character of the climbing fiber influence. Marr uses only data from Eccles et al indicating that climbing fibers are powerfully excitatory. On this basis, Marr postulates that climbing fibers affect learning through strengthening of parallel fiber synapses on Purkinje dendrites.

In contrast, Albus includes additional data from other sources indicating that climbing fiber effects are much more complex.

"Each Purkinje cell is contacted by a single climbing fiber. In a conscious animal the climbing fibers fire in short bursts of one or more spikes at a rate of about 2 bursts/sec [5, 18]. Each climbing fiber burst causes a single spike on the Purkinje axon followed by a complex burst of spike-like activity in the Purkinje dendritic tree and intense depolarization of the Purkinje cell. The single axon spike is followed by a pause in the spontaneous Purkinje axon spike activity for 15-30 msec. This pause, accompanied by intense depolarization, was first observed by Granit and Phillips [8] and was termed the inactivation response to distinguish it from a normal pause in activity resulting from hyperpolarization. After the 15 to 30 msec inactivation response, the cell gradually recovers its spontaneous firing rate over a period of 100-300 msec [3]. As it approaches normal, the cell becomes once again responsive to parallel fiber input activity." [From Albus 1971]

On the basis of this data, Albus suggests that the primary effect of climbing fiber input is to cause the Purkinje to pause, i.e. the net results is inhibitory, despite the initial excitatory spike. He further hypothesizes that climbing fibers effect learning through weakening parallel fiber synapses, not only on Purkinje dendrites, but on nearby Basket and Stellate cells as well.

This is a counterintuitive idea which not only disagrees with Marr's theory of synaptic facilitation, but with virtually the entire tradition of neurophysiological and psychological learning theory. Almost without exception, previous theories had been influenced by the Pavlov, Hebb, Skinner presumption that learning occurs by facilitation of synapses due to their association with behavior leading to successful results; not by synapses being weakened by contributing to unsuccessful behavioral results. In fact, the entire branch of psychology founded by Skinner has generalized this notion to the point of opposing the principle of teaching by punishing incorrect behavior.

The notion of learning from error correction (i.e. weakening synaptic weights that contribute to undesirable results) comes from engineering. It is the fundamental principle of servomechanisms (i.e. negative feedback of an error signal). It was put into a neurological context by the Perceptron and its derivatives such as the Adeline[Wid85], the Cerebellar Model Articulation Controller (CMAC)[Alb75], and neural nets." [Hop82, Gro75].

Albus suggests as a possible mechanism for synaptic weakening that there exists a critical interval near the end of the inactivation response after the effect of the climbing fiber burst has worn off sufficiently so that the cell can be fired by parallel fiber input but before the dendritic membrane has returned completely to normal. If the Purkinje cell fires in this interval, this firing is an error signal that signals every active parallel fiber synapse to be weakened.

The amount of weakening of each synapse is proportional to how strongly that synapse is exciting the Purkinje cell at the time of error signal. The effect of this mechanism would be to train the Purkinje cell to pause at the proper times, that is, at climbing fiber burst times. After learning is complete, the Purkinje knows when to pause because it recognizes the mossy-parallel fiber pattern that occurred previously at the same time as the climbing fiber burst. Later, since each parallel fiber active synapse was weakened by the error signal, if the same mossy-parallel fiber pattern occurs again, the Purkinje will pause even without the climbing fiber burst. Thus, the Purkinje is forced to perform in a certain way by the climbing fiber teacher. After learning is complete, it behaves in that same way, under the same mossy fiber conditions, even in the teacher's absence. [Alb71]

Albus goes on to hypothesize that synaptic weakening also occurs at the parallel fiber synapses on Basket and Stellate b dendrites. This effectively provides both positive and negative training adjustments. Positive adjustments occur by weakening excitatory synapses on inhibitory interneurons, and negative adjustments by weakening excitatory synapses on the Purkinje output cells.

Albus argues that synaptic weakening is necessary as a learning mechanism for precise motor learning, because otherwise synapses quickly become saturated.

If a synaptic weight is increased each time it correctly fires, repeated learning will eventually cause it to saturate. This means that continued training in motor skills will produce degraded performance.

"Yet, it is an obvious fact that continued training in motor skills improves performance. Extended practice improves dexterity and the ability to make fine discriminations and subtle movements. This fact strongly indicates that learning has no appreciable tendency to saturate with overlearning. Rather, learning appears to asymptotically approach some ideal value. This asymptotic property of learning implies that the amount of change that takes place in the nervous system is proportional to the difference between actual performance and desired performance. A difference function in turn implies error correction, which requires a decrease in excitation upon conditions of incorrect firings."[Alb71]

Conclusions

Recent experimental data confirms the basic Marr-Albus hypothesis in three important respects:

1) motor learning does indeed occur in the cerebellum,
2) parallel fiber synapses on the Purkinje dendrites are modified, and
3) the modification is produced by concurrent activity of climbing fibers. [Ito84].

It has also been shown experimentally that cerebellar learning is accomplished through weakening of variable synapses, as predicted by Albus alone [Ito84]. Observations of negative as well as positive changes in synaptic strength have also been observed in the visual cortex [Rui69,Ros72]

Thus, the Marr and Albus theories have become two of the best working hypotheses currently available to cerebellar researchers.

Both the Marr and Albus theories make a number of additional predictions about neuronal function in the cerebellum, as well as the relationship between the cerebellum and other centers of motor control. These have not yet been either confirmed or disproven by experimental evidence. For example, there is as yet no evidence that the responsiveness of a basket cell to mossy fiber inputs is modified following conjunctive activation of the mossy fibers with climbing fibers. [Ito82]

In other areas, the CMAC model based on the Albus cerebellar theory is now being used to perform dynamic computations for fine motor control of robot arms [Alb75, Mil87]. A control system architecture based on CMAC principals has been used for the control of automated manufacturing facilities [Alb81], for controlling Multiple Autonomous Undersea Vehicles [Alb88], and will be implemented on the Flight Telerobotic Servicer [Alb87] being built for the NASA Space Station.

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